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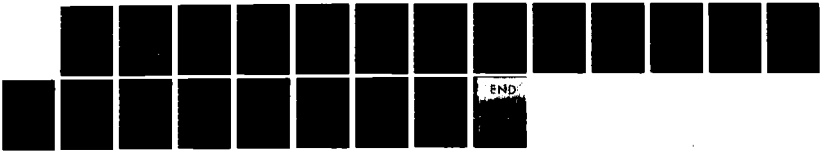

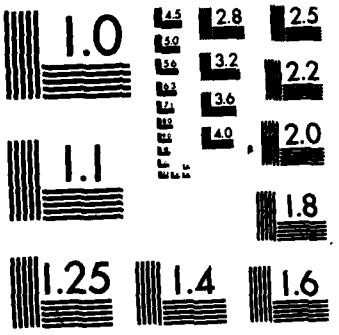

BIOCHEMICAL RESPONSES OF NAVY SPECIAL WARFARE PERSONNEL TO CARBOHYDRATE LOADING AND PHYSICAL PERFORMANCE

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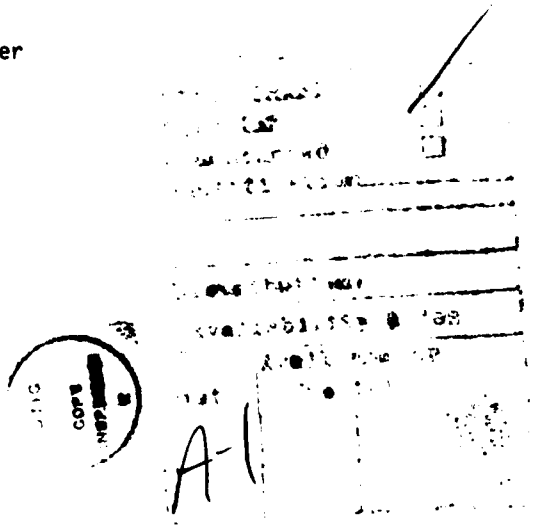

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SUMMARY

Carbohydrate loading, a program of diet and exercise modification, has been shown to increase muscle glycogen stores and increase endurance capacity at work rates above 60-70% of maximal aerobic capacity. Such endurance enhancement might be of value to Navy special warfare personnel for certain high-risk missions. Therefore, the effectiveness of a program of carbohydrate loading was tested on a sample of special warfare personnel.

In conjunction with this test, selected biochemicals were measured during the program, during a control, non-loading program, during performance tests subsequent to each program to monitor the safety of the carbohydrate loading program, and to get indications of the metabolic changes accompanying the program.

The following serum biochemistries were measured: the enzymes creatinine phosphokinase (CPK), lactate dehydrogenase (LDH), and 2-hydroxybutyrate dehydrogenase (HBD) to assess potential muscle injury and cardiovascular stress; sodium and potassium to assess electrolyte shifts; total protein and albumin concentrations to assess alteration in hydration levels; glucose and cortisol to document shifts in carbohydrate metabolism; and uric acid and creatinine concentrations as additional indicators of muscle strain.

Changes in glucose and cortisol during the carbohydrate loading program were consistent with expected metabolic shifts during the dietary alterations. The differences in other biochemical responses to the two test programs tended to reflect the difference in exercise levels between them. The biochemistries changed in the expected fashion during the endurance tests. None of the responses suggested muscle or cardiovascular pathology. Elevation of HBD following the carbohydrate loading program is consistent with a lack of long distance endurance training in the current special warfare program, and suggests a need to explore further the interactions among activity duration, work rate, physical fitness, and response to ergogenic aids such as carbohydrate loading.

INTRODUCTION

There are special groups of forces within the military whose missions may involve high physical workloads, often for extended periods of time under conditions of uncertain food supply and irregular sleep intervals. Such forces within the Department of the Navy are the Marine Reconnaissance Battalions, the Navy Underwater Demolition Team (UDT), and Sea, Air, and Land (SEAL) team personnel. For these forces, chances of mission success are improved by enhancing endurance capacity. One method of such enhancement is by a program of diet and exercise modification called "carbohydrate loading."

At moderately high workloads (above 60-70% of maximal aerobic capacity) the limitation to continued physical performance is the exhaustion of the stores of glycogen contained within the muscle cells (1,2). Following a carbohydrate loading program can increase the muscle glycogen stores to approximately twice their normal levels (3-5). Carbohydrate loading consists of two phases, a glycogen stores depletion phase and a repletion or loading phase. During the depletion phase, the individual performs long-duration, moderately intense exercise while consuming a diet low in carbohydrate content. This regimen depletes the muscle glycogen supplies and presumably stimulates the mechanisms responsible for synthesis and storage of glycogen (5). During the subsequent loading phase, the individual performs minimal exercise while consuming a diet high in carbohydrate. Because the glycogen synthesis and storage mechanisms are "activated," there is a rebound hyper-repletion of the glycogen stores.

Although there has been documentation of the effectiveness of carbohydrate loading both in terms of performance changes (6,7) and muscle glycogen changes (3-5) following such programs, the metabolic processes which might be occurring consequent to carbohydrate loading are poorly documented. Therefore as a part of a pilot study to test the effectiveness of a carbohydrate loading program on a group of special warfare personnel, we monitored a set of serum biochemicals during both the imposition of a carbohydrate loading program and the subsequent endurance test.

Biochemistries were selected to monitor muscle damage or fatigue, and to provide indications of the shifts between primary fuel substrates. The enzymes creatinine phosphokinase (CPK), and lactate dehydrogenase (LDH), were measured as indicators of relative muscle damage (8, p. 653, 683; 9). 2-hydroxybutyrate dehydrogenase (HBD) was measured to estimate stress to the cardiovascular system. HBD activity represents the activity of the LDH derived from the heart and red blood cells, the so-called LD-1 and LD-2 isoenzymes of LDH (8, p. 599). Most of the remainder of the LDH released during exercise is derived from skeletal muscle. Monitoring HBD activity in addition to total LDH allows us to estimate the proportion of LDH released from the heart and red blood cells separately from that released from skeletal muscle. Uric acid and creatinine levels were measured as additional indicators of muscle injury (11). Uric acid also indicates changes in purine metabolism.

Sodium and potassium levels were monitored to determine whether or not major shifts in electrolyte balance occurred as a result of carbohydrate loading. Serum total protein and albumin concentrations were monitored to be sure there were no hydration alterations resulting from the diet/exercise program.

Levels of serum glucose and cortisol were monitored to document the shifts in

carbohydrate metabolism accompanying the loading program. In addition, each participant's urine was checked for the presence of ketones to monitor shifts to fat metabolism.

METHODS

Participants

The participants in this study were 9 male UDT and SEAL team personnel, aged 22-36 years and attached to Special Warfare Group One, Coronado, CA. The participants represented a cross-section of the special warfare group in their normal readiness state. Each participant was briefed on the nature of the study and the risks involved in participation in the study. Each participant gave his voluntary consent to participate with the understanding that he could withdraw from the study at any time. Prior to his acceptance into the study, each participant filled out a medical history and passed a physical examination.

Twenty-four special warfare personnel originally volunteered to participate in this study. However, 15 of these original participants were eliminated from the study because of failure to comply with the diet or exercise restrictions, transfer to other units, or illness. Characteristics of the 9 participants remaining in this study are presented in Table 1.

Procedures

Maximal rate of oxygen consumption ($\dot{V}O_2$ max) was determined for each participant from open-circuit spirometry measures taken during an interrupted treadmill test (12). Participants were rank-ordered on their individual $\dot{V}O_2$ max values and alternately assigned to one of two experimental groups. Endurance performance was measured for individuals in each group after following a six-day carbohydrate loading program and after following a six-day non-loading regimen. The order of presentation of the two programs was reversed for the two groups.

Diet/Exercise Programs

Following the suggestions of Astrand (13) and Karlsson and Saltin (7), a six-day loading program consisting of three days of glycogen depletion and three days of glycogen repletion was used. The program is described in Table 2. The 14-mile depletion run represents 3.5 times the normal training distance of these participants. The control, non-loading program is described in Table 3.

Food for the participants was provided by the investigators. Three diets were utilized: a diet high in carbohydrate content (high-CHO); a diet low in carbohydrate content (low-CHO); and a diet of normal composition (normal) (14). Each diet consisted of a combination of solid food and liquid formula and provided approximately 3500 kilocalories per day. The amount and kind of solid food was the same for all diets. The proportions of the constituents of the liquid formula varied with each particular diet. Composition of the three different diets is given in Table 4.

Endurance Measurement

Endurance performance was measured as the length of time a participant could run on a motor-driven treadmill at 0% grade and at a speed requiring him to work at approximately 80% of his VO_2 max. The endurance run was conducted in an interrupted fashion. The participant would run for 18 minutes and would then be allowed a 2 min rest. Water was provided *ad libitum* during the run. Electrocardiogram, heart rate, and rectal temperature

were monitored throughout the run. Near the mid-point of each 18-min running period, $\dot{V}O_2$ was determined, and if necessary, the treadmill speed was adjusted to maintain a work load of approximately 80% $\dot{V}O_2$ max. The test was terminated when the participant indicated he could no longer continue running at this work load. For the second endurance test, the speed/time profile of the first test was repeated.

TABLE 1

CHARACTERISTICS OF PARTICIPANTS

PARTICIPANT NUMBER	AGE (yr)	HEIGHT (cm)	WEIGHT (kg)	% BODY FAT	$\dot{V}O_2$ MAX (l/min)	$\dot{V}O_2$ MAX (ml/kg · min)
Group I						
06	32	175.3	76.5	14.9	5.43	71.11
22	36	182.9	83.5	18.2	4.47	53.58
28	30	170.2	60.8	12.6	3.19	52.43
42	25	172.7	79.4	16.0	4.93	62.03
86	30	182.9	71.7	12.1	4.73	66.01
$\bar{X}$	30.6	176.8	74.4	14.8	4.55	61.01
SD	(4.0)	(5.9)	(8.7)	(2.5)	(0.84)	(8.04)
Group II						
11	25	182.9	74.9	10.8	4.30	70.73
51	22	188.0	79.9	6.7	4.19	52.46
69	30	182.6	67.6	16.7	4.31	63.66
80	26	170.2	64.0	10.7	4.19	65.45
$\bar{X}$	25.8	175.9	71.6	11.2	4.50	63.08
SD	(3.3)	(11.6)	(7.2)	(4.1)	(0.54)	(7.69)
Sample $\bar{X}$	28	176.4	73.1	13.2	4.53	61.93
SD	(4.3)	(8.2)	(7.7)	(3.6)	(0.68)	(7.46)

TABLE 2

CARBOHYDRATE LOADING DIET/EXERCISE PROGRAM

	DAY	DIET	EXERCISE
Depletion Phase	1	Low CHO	14-mile run*
	2	Low CHO	6-mile run
	3	Low CHO	4-mile run
Loading Phase	4	Hi CHO	1-mile run
	5	Hi CHO	1-mile run
	6	Normal	No run
Test	7	Normal	Endurance run

**Average running time = 113 min.*

TABLE 3

NON-LOADING DIET/EXERCISE PROGRAM

	DAY	DIET	EXERCISE
	1	Normal	4-mile run
	2	Normal	4-mile run
	3	Normal	4-mile run
	4	Normal	4-mile run
	5	Normal	4-mile run
	6	Normal	No run
Test	7	Normal	Endurance run

TABLE 4

DIET COMPOSITION

Solid Food:*	Liquid Formula:**	Fluids:
194g water-packed tuna	Calcium caseinate	Water, diet soda, and coffee provided *ad libitum*
142g canned chicken	Corn oil	
2 hard-boiled eggs	Fructose	
50g mayonnaise	Polycose®	
Lettuce *(ad libitum)*	Minerals, flavorings and saccharin	
	Approximately 1.9 liters water	

**Constant for all diets*
***Proportions varied with treatment diet*

TREATMENT DIETS

% CALORIES FROM:	LOW CHO DIET	HIGH CHO DIET	NORMAL DIET
CHO	3	64	46
Fat	50	24	42
Protein	47	12	12
Total Calories = 3500/day			

Biochemical Measurement

Biochemistries were determined from the serum of 10-ml blood samples obtained by venipuncture of a superficial vein of the anticubital fossa. Fasting blood samples were collected each morning of the diet/exercise program from a random subset of the participant sample. Additionally blood samples were collected four times on the day of each endurance run: immediately prior to beginning the run; after having run for 60 min; immediately upon completion of the run; and 60 min after having completed the run. As indicated, serum samples were analysed for Na^+; K^+; glucose, creatinine; uric acid; cortisol; the enzymes CPK, LDH, and HBD; as well as albumin and total protein.

Sodium and potassium were determined by emission flame photometry (15, pp. 873-879); glucose by the orthotoluidine method (16, pp. 249-251); creatinine by the Jaffe reaction (Pierce Chemical Co., Rockford, IL); and uric acid by production of tungsten blue from reaction with phosphotungstic acid (American Monitor Corp., Indianapolis, IN). Cortisol was determined by radioimmunoassay (Miles Laboratory Inc., Elkhart, IN). The enzymes LDH, CPK, and HBD were determined spectrophotometrically utilizing commercially available kits (Worthington Biochemicals Corp., Freehold, NJ). Albumin was measured by bromcresol green binding (Pierce Chemical Co., Rockford, IL), and total protein by the biuret method (17, pp. 302-304).

Morning urine samples were tested for the presence or absence of ketone bodies using Ketostix™.

Analysis

Due to the attrition of participants from the study, only portions of the biochemical data could be analysed statistically. Fasting samples collected during the diet exercise programs did not provide sufficient sample numbers at each collection to allow analysis. The data collected during the endurance runs were analysed using a two-way within-person analysis of variance (18) with collection time in the run as one treatment and form of the diet/exercise program (load vs. non-load) as the other treatment. Because this study was intended as a pilot for further work, the level for significance was set at $p=0.05$. Furthermore, marginal significance was recorded whenever $p<0.1$, since at this level and with this sample size (9 subjects) the power of the analysis was 0.5 for large differences (25). In that some of the parameters that we were measuring gave indications of the relative safety of the carbohydrate loading procedure, we wanted to minimize the possibility of missing meaningful effects because of our small sample size.

RESULTS

Results of the endurance runs following each of the programs have been reported previously (19). In summary, participants had a 9% increase in running time (10.8 min) following the carbohydrate loading program over that following the non-loading program.

Graphical representations of the mean values at each collection time for each of the biochemicals measured are provided in Figures 1-6.

As expected, serum glucose (Figure 2) decreased during the depletion phase of the carbohydrate loading program. (Note, the blood sample for day 4, the first day of repletion, is taken before the first high-CHO meal was ingested.) The glucose level increased following the start of the loading phase, although apparently only transiently. Furthermore, serum cortisol (Figure 4) concentrations appeared elevated by the morning of

day 4, but had returned to day 1 values by day 7.

The enzymes LDH, CPK (Figure 5), and HBD were all elevated during the depletion phase of the carbohydrate loading program. Levels of LDH and CPK had returned to day 1 values by day 7, but HBD remained elevated.

Results of the urine analysis were that all participants showed ketouria by the third day of depletion on the carbohydrate loading program but no participants evidenced ketouria after the first day of change to the high-CHO diet. No ketouria was seen with the non-loading diet.

Levels of all of the biochemical parameters measured, save K^+ changed significantly during the endurance run, although for Na^+ this significance was only marginal ($F_{3,21}=2.82$, $p=0.064$. For all others $p<0.004$).

Significant effects of the load were detected for uric acid concentrations ($F_{1,8}=9.305$, $p=0.016$) which were generally lower following the loading program than following the non-loading one (see Figure 3); and for HBD concentrations ($F_{1,8}=8.07$, $p=0.022$) which were generally higher following the loading program (see Figure 6).

Significant interactions (differences in the time response patterns between diet/exercise programs) were found for LDH ($F_{3,24}=4.314$, $p=0.014$) and albumin ($F_{3,24}=3.933$, $p=0.020$) and marginally significant interactions were found for uric acid ($F_{3,24}=2.892$, $p=0.056$) and CPK ($F_{3,24}=2.793$, $p=0.062$). However in no case did the post hoc pairwise comparisons (Newman-Keuls procedure; 18, pp. 91-93) yield significant simple main effects.

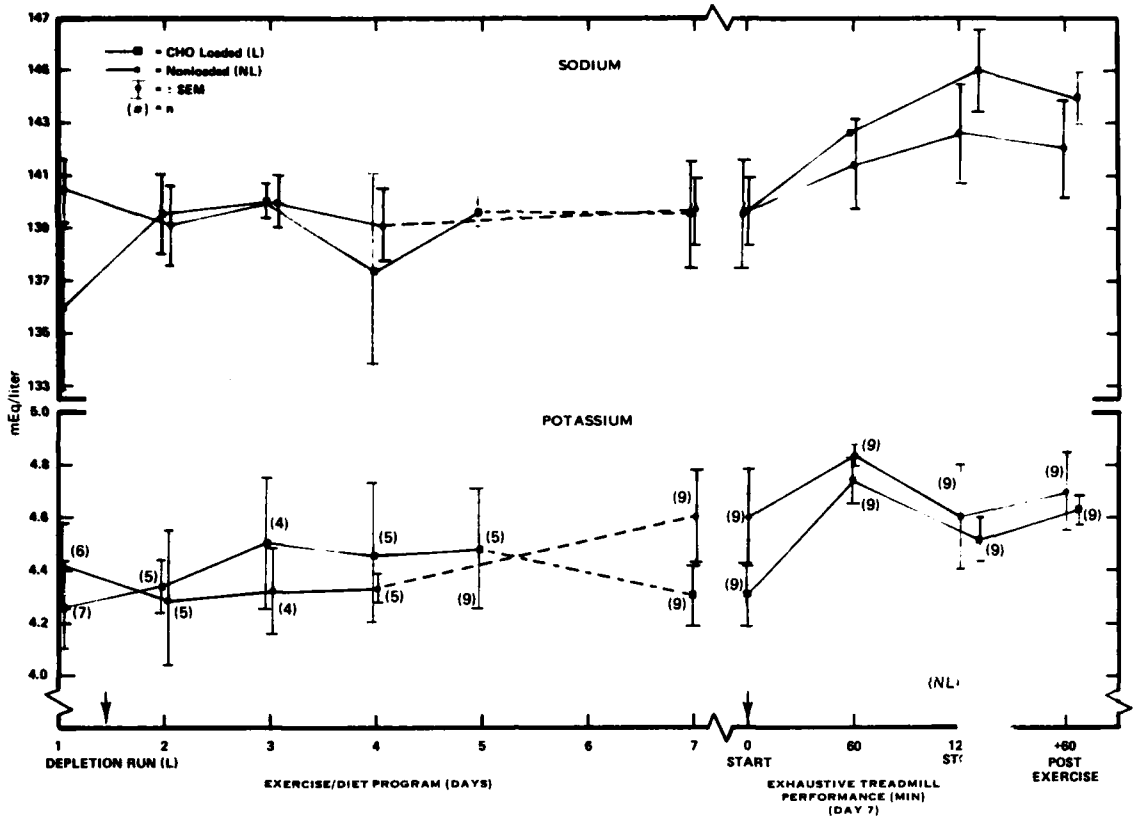

Figure 1. Serum Potassium and Sodium Response

NOTE: Mean values are shown with the standard error of the mean. Number in parentheses indicates sample size for determining that mean value. Two time scales are provided. The left-hand portion shows the mean values of the fasting samples obtained on days 1–7 of each program; the right-hand portion, the mean values of the samples obtained during the endurance runs. The stop and 60-min post exercise points are offset to reflect the difference in mean run times for the two programs. Points indicated as "day 7" are identical to those indicated as "time 0" of the endurance run.

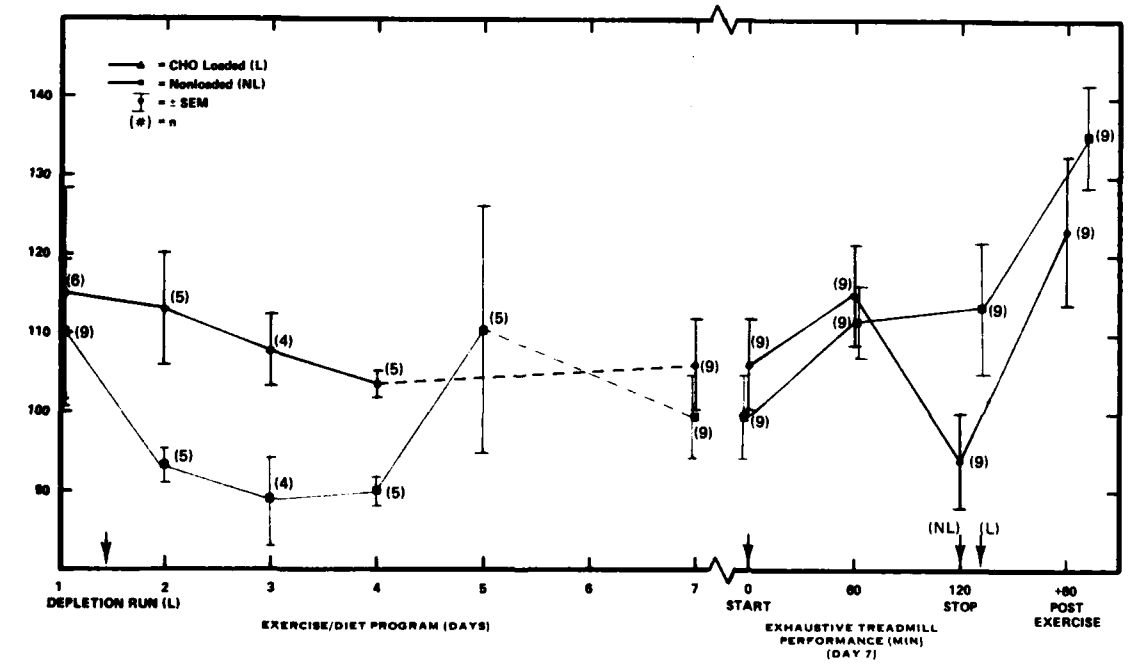

Figure 2. Serum Glucose Response

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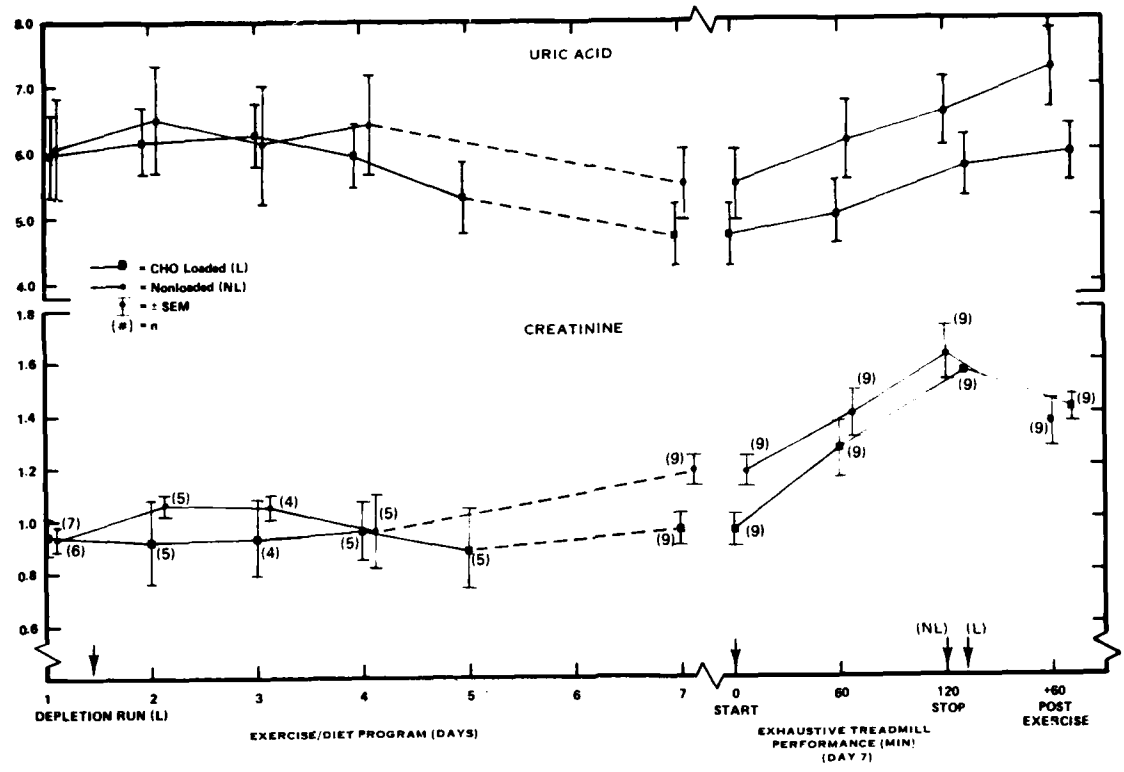

Figure 3. Serum Creatinine and Uric Acid Response.

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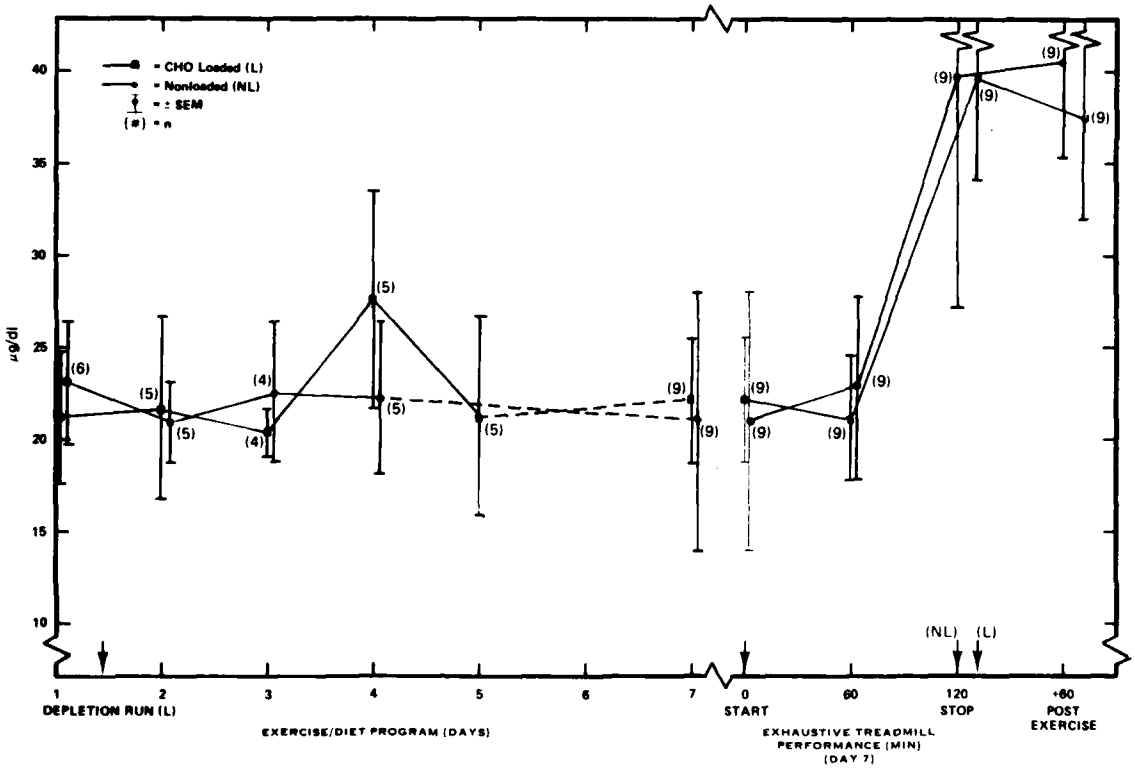

Figure 4. Serum Cortisol Response.

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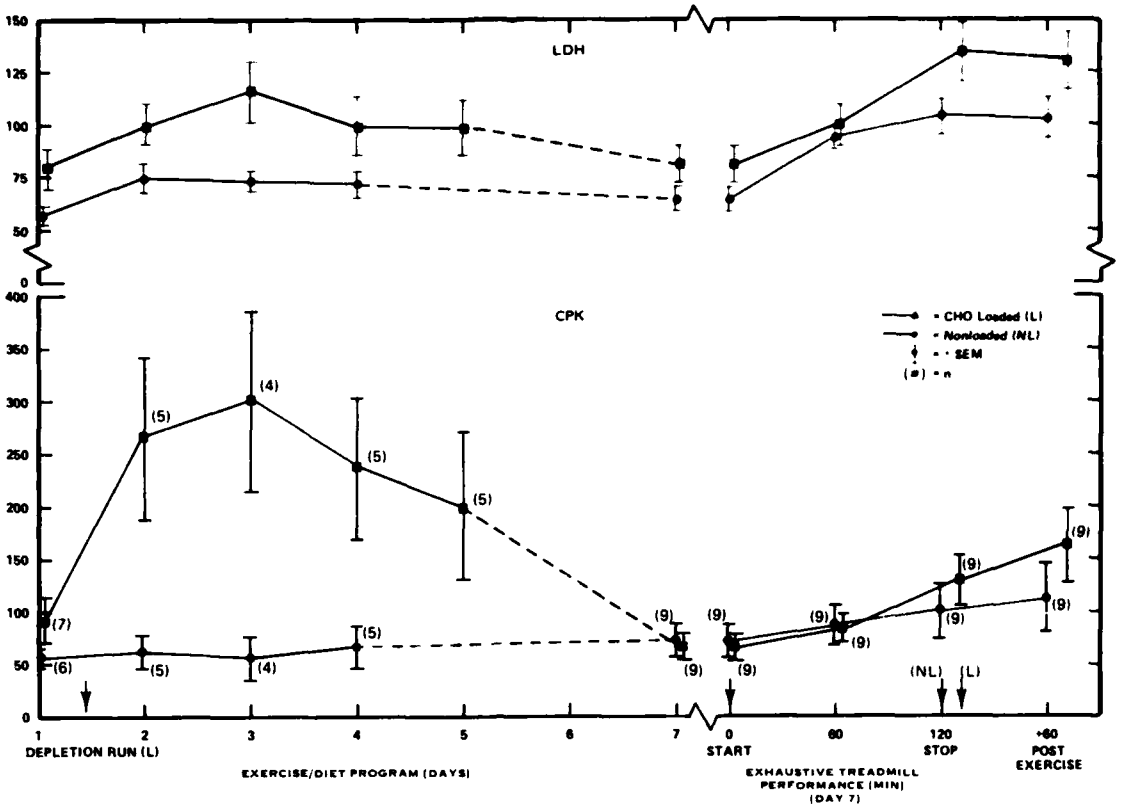

Figure 5. Serum CPK and LDH Response.

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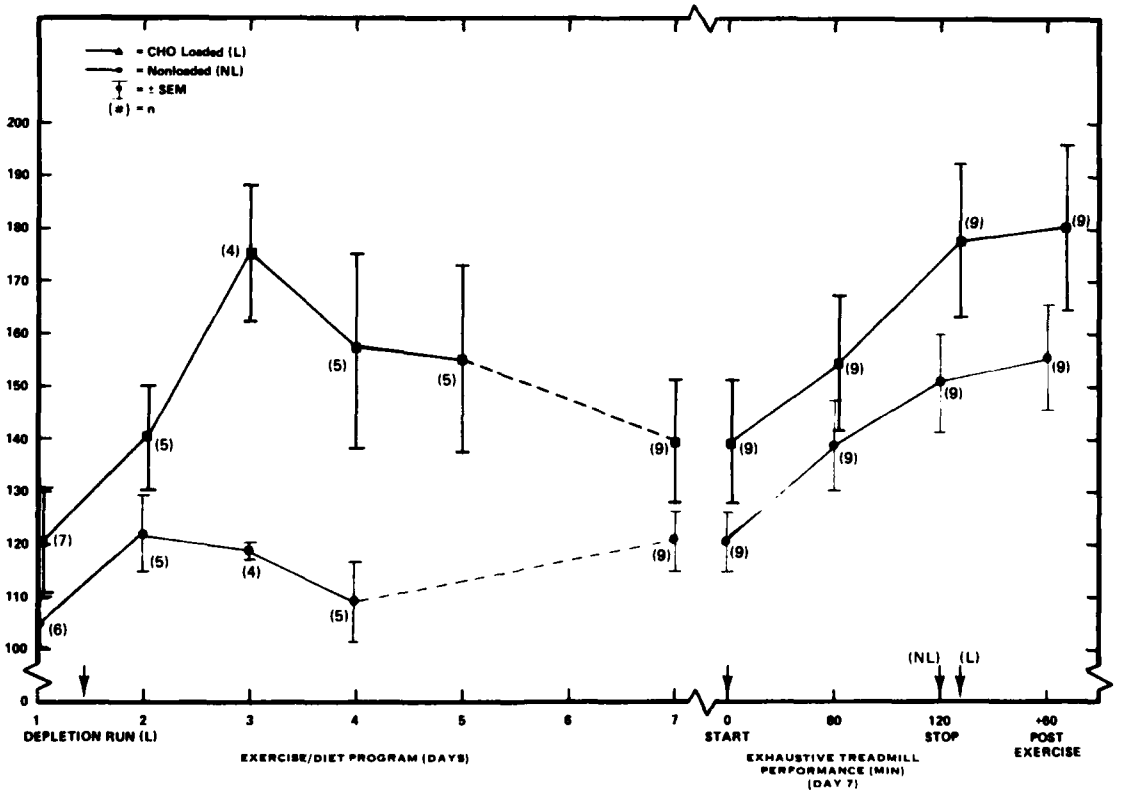

Figure 6. Serum 2-hydroxybutyrate Dehydrogenase Response.

See note, Figure 1 for explanation of figure organization.

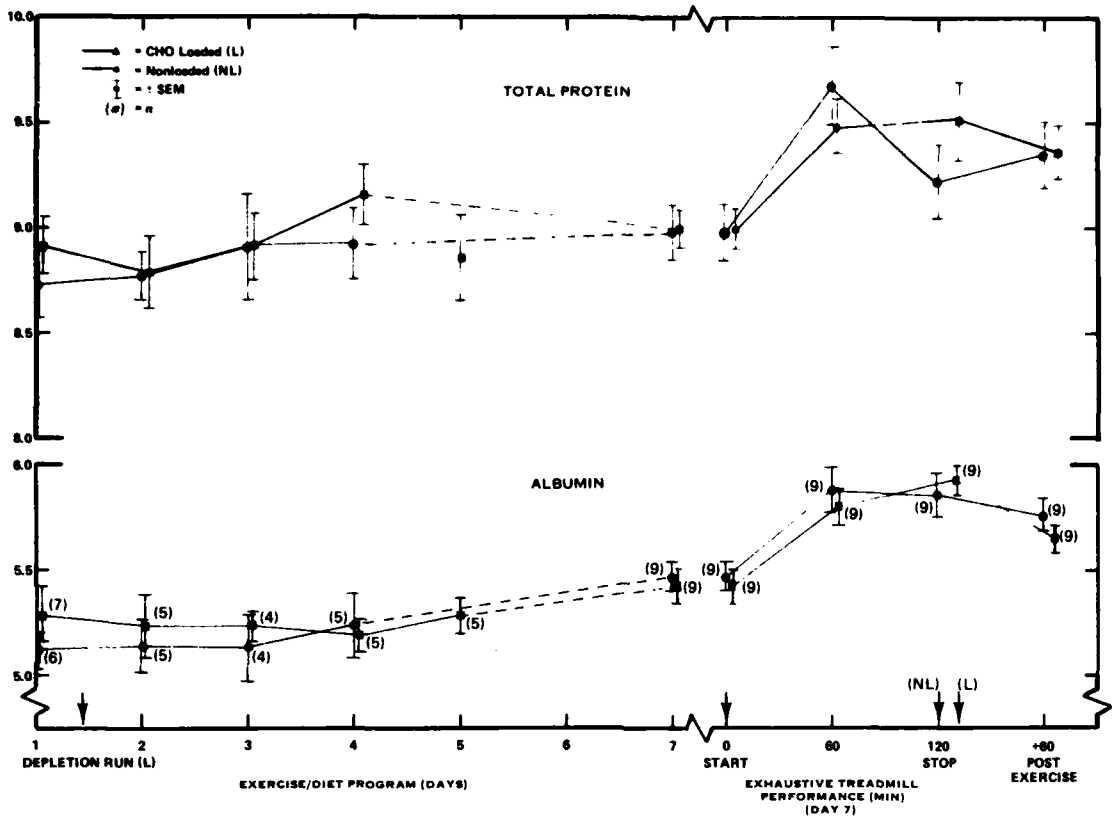

Figure 7. Serum Albumin and Total Protein Response.

See note, Figure 1, for explanation of figure organization.

DISCUSSION

The results of this study did not reveal any major risks associated with carbohydrate loading. There appear to be no changes in Na^+ or K^+ during either diet/exercise program. The modest increase in Na^+ seen during the endurance runs is consistent with previous findings of other workers (4,11). Furthermore, comparison of the albumin and total protein patterns between the two programs suggest no major differences in hydration levels during either the program or the subsequent endurance test, although the significant interaction term for albumin may represent a slight protein-sparing effect of the carbohydrate load at the end of the endurance run.

The serum glucose depression during the depletion phase of the carbohydrate loading program coupled with the increase in serum cortisol suggest that glycogen stores were truly depleted, at least in the liver, and gluconeogenisis had been stimulated. Although the patterns of glucose response during the endurance runs did not differ significantly, it is noteworthy that the serum glucose is noticeably lower at the end of the endurance run following the non-loading program than at the end of the run following the loading program. This finding is in keeping with the results of Hultman and Nilsson (30) who find that liver glycogen levels are also elevated by carbohydrate loading and those of Ahlborg and his co-workers (21) who find work capacity limits to be related to muscle glycogen levels rather than blood glucose levels.

The temporal changes in CPK and LDH durig the loading program appear to be due to the prolonged exercise associated with the depletion phase. The concentrations measured and the time course for their elevation match values reported by others for this exercise intensity (22). The increases in CPK and LDH concentrations measured during the endurance runs were also similar to those reported by other investigators (10). At no time were serum concentrations of CPK or LDH seen which might be indicative of severe muscle damage. Such levels are often 6-10 times greater than those seen following vigorous exercise (23).

The HBD response to carbohydrate loading may also reflect the effects of the increased exercise associated with carbohydrate loading. However interaction with dietary effects cannot be ruled out. Rose and his co-workers (26,27) and Stromme and his co-workers (22) have looked at LDH isoenzymes in trained endurance runners prior to and following races ranging from 10-70 km. In each study LD-1 and LD-2 fractions were unchanged following the exercise, the majority of the change in total LDH apparently due to changes in LD-5, the skeletal muscle and liver fraction. However as Rose points out (26), the lack of LD-1 and LD-2 response may reflect the level of endurance conditioning of their participants. Work with rats (28,29) indicates that LD-1 and LD-2 are elevated in untrained rats following exercise. However, following a period of endurance training, the exercise induced changes in these isoenzymes decreases with training. These decreases seem to be a function of the duration of the training sessions (29). Therefore, one interpretation of our findings is that these UDT and SEAL team personnel are not highly trained for long-distance running. The continued elevation of HBD during the repletion phase of the program is consistent with clinical reports that HBD activity has a long half life compared to other enzymes such as CPK or other LDH isoenzymes (31).

Just as was the case with CPK and total LDH, serum concentrations of HBD were elevated above normal values (31) following the depletion run. But again these values were not as great as those seen following a major pathology such as a myocardial infarct (31).

Our HBD data do not allow us to determine the source of the HBD activity with certainty. It may likely derive from increased myocardial activity or the destruction of red blood cells (8, p. 599). To our knowledge, this LDH isoenzyme pattern has not been previously documented for exercising humans. These findings point to a need for further exploration not only of the effects of training on LDH exercise response but also of the effects of exercise on the cardiovascular system and the mechanisms that underlie the release of enzymes from various tissues.

Interpretation of the endurance run interaction results for LDH and CPK is difficult. The CPK interaction is of marginal significance and may not be meaningful. In the absence of a significant interaction for HBD ($F_{3,24}$=1.53, p=0.23), the observed interaction for LDH suggests the release of muscle LDH at the end of the endurance run. Taken with the marginal CPK results, the possibility of increased muscle strain while running with "loaded" muscles cannot be ruled out. However, work by Pate and his co-workers (10) suggests changes which take place with increasing exercise duration at a fixed exercise intensity are not linear. Therefore from this sample it cannot be determined whether the differential increases in enzyme concentrations following the different programs are a normal concomitant of the increased running time following the loading program or represent increased muscle strain.

The decrease in serum uric acid concentration following carbohydrate loading is consistent with previous reports that a high carbohydrate diet tends to increase uric acid secretion (20). The mechanism for this effect is unclear. The decrease may also be a result of the decreased exercise level during the loading phase of the program (11).

One must bear in mind that with this sample size, the loading effects and loading time interactions must be viewed cautiously. There is a clear need for replication of these findings. There were no major changes in electrolytes, and no unexpected changes in serum protein or albumin values. The CPK and LDH results do not suggest muscle cell damage. However, the HBD results do suggest a need to investigate the relationship between endurance conditioning and the physiological/biochemical responses to this program. Additionally, possible effects of increasing the rate of carbohydrate metabolism on fatigue processes at different energy expenditure rates warrant further consideration. Such effects will determine whether or not carbohydrate loading is advisable for situations involving work rates which only infrequently exceed 60-70% of maximal capacity.

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Selected biochemistries were monitored in 9 Navy Special Warfare personnel during a program of carbohydrate loading (LOAD), during a control, non-loading program (NONLOAD), and during an endurance test subsequent to each program. Each program required 6 days with the endurance test on the 7th. The endurance test consisted of interrupted running (18 min. running/2 min. rest) to voluntary exhaustion on a treadmill at a speed requiring an energy expenditure of about 80% of maximal aerobic capacity. Fasting blood samples

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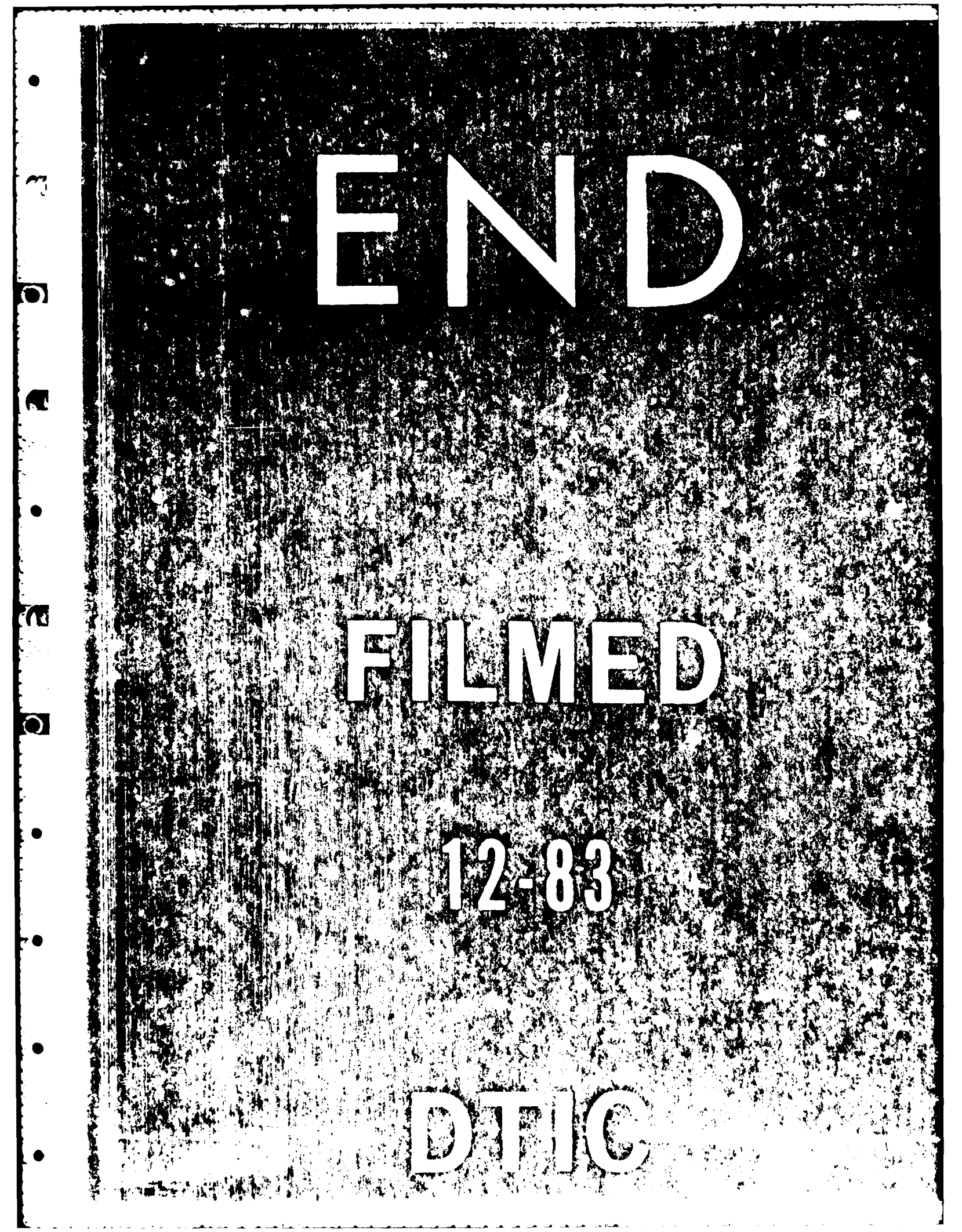
20. Abstract (continued)

were collected each morning of the diet/exercise programs, and samples were collected prior to, 60 min. into, at the end of, and 60 min. after each endurance test. Blood samples were analyzed for the enzymes creatine phosphokinase (CPK), lactate dehydrogenase (LDH), and 2-hydroxybutyrate dehydrogenase (HBD); sodium; potassium; total protein; albumin; glucose; cortisol; uric acid; and creatinine. In addition, urine samples were checked each day for the presence of ketones. When compared to the NONLOAD results, CPK, LDH, and HBD were all elevated during the LOAD depletion phase, reflecting the greater exercise demands. Blood glucose decreased during depletion and cortisol was elevated as expected with carbohydrate deprivation. During the LOAD repletion phase, CPK, LDH, and HBD values decreased, although only CPK achieved pre-LOAD levels. Uric acid also decreased. All other blood parameters showed no differences between LOAD and NONLOAD. Urine analysis revealed ketouria by the end of LOAD depletion in all participants. In all cases, ketouria was ameliorated by one day of repletion. All biochemical values were increased during the endurance tests except potassium. There appeared to be a slightly greater increase in muscle LDH and in CPK following load, which may have been associated with the longer endurance time following LOAD. In no case did the enzyme values suggest major muscle or cardiovascular damage. HBD was elevated following LOAD, suggesting a lack of long-distance running training in the Special Warfare training program. This HBD elevation was taken to suggest a need to explore further the effects of endurance running in nondistance trained individuals. Additionally, a need is suggested to explore the effects of physical fitness parameters on the effectiveness of ergogenic programs such as LOAD.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